resolution of PET to define the precise locations of ^{18}F-NaF uptake in coronary arteries.

Finally, we do agree with Dr. Mohler and colleagues that, after further validation, ^{18}F-NaF PET of the coronary arteries may give unique insights into the pathophysiology of calcium deposition and perhaps its dispersal with appropriate therapy.

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Efficiently Doing the Wrong Thing

We would like to commend Felker and Mentz (1) for their comprehensive review of volume removal therapy for patients with acute decompensated heart failure (ADHF). However, we also would like to challenge their most basic assertion that "Fluid retention and congestion are responsible for 90% of HF hospitalizations." Neither of the references they cite (2,3) provide data to support that fluid retention occurs before the onset of ADHF in most patients. In fact, more recent studies that have measured weight gain (as a surrogate for fluid retention) show clearly that most patients gain either no, or minimal, weight before hospitalization for ADHF (4,5) despite increased cardiac filling pressures (5).

To account for increased cardiac filling pressures in the absence of weight gain, volume shifts rather than volume gains must occur. In a recent manuscript (6), we elucidate the likely mechanisms underlying these shifts, which involve a normally compliant splanchnic venous system that becomes noncompliant and results in redistribution of volume to the cardiopulmonary circulation, a process that can occur rapidly. We also point out that even in the minority of patients who do experience weight gain, little of the excess fluid resides within the effective circulatory volume (approximately 2.5%). The majority of patients presenting with congestion likely have a syndrome of sympathetically mediated redistribution of volume and diuretic resistance.

Fluid retention and congestion do not necessarily represent the same phenomenon, and congestion most often is not due to fluid overload. Thus, strategies aimed at removing salt and water will necessarily be fraught with complications, including intravascular volume depletion, activation of the renin-angiotensin system, worsening renal function, and iatrogenically induced cardiorenal syndrome. Therefore, we caution readers to reconsider volume removal as the prime target of therapy for patients with ADHF. Diagnosing the contribution of redistribution versus total body salt and water gain emerges as the primary decision point, followed by therapy directed at the underlying etiology of congestion.

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Reply

We thank Drs. Dunlap and Sobotka for their interest in our recent review on volume removal in patients with acute decompensated heart failure (ADHF). We agree with them that redistribution of volume (rather than increase in total body volume) may be an important and underappreciated mechanism in patients with acute decompensated heart failure, as our group has suggested previously (1).

Although we welcome "outside the box" thinking in a field that clearly would benefit from new ideas, we also suggest that the results from recent clinical trials might dampen the enthusiasm for

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the hypothesis that redistribution of volume is the primary pathophysiology of most patients with ADHF. First, if redistribution were the primary culprit of decompensation, it would follow that vasodilator therapy would be a highly effective therapy for ADHF. Unfortunately, the largest vasodilator trial ever performed in heart failure, the recently completed ASCEND-HF (Acute Study of Clinical Effectiveness of Nesiritide in Decompensated Heart Failure) study, showed no significant clinical benefit of vasodilator therapy (2). Secondly, the randomized, double-blinded DOSE (Diuretic Optimization Strategies Evaluation) study suggested greater efficacy in terms of symptom relief and decongestion with a high-dose versus a low-dose loop diuretic regimen, without any evidence for long-term harm on renal function or clinical endpoints (3). These data would seem to contradict the assertion that excess volume is not an important mechanism for most patients with ADHF and that "strategies aimed at removing salt and water will necessarily be fraught with complications."

Although both volume overload and volume redistribution are likely to be operative in individual patients to varying degrees, current diagnostic tools to not appear adequate to distinguish these mechanisms clinically. We strongly agree with the general suggestion in the final sentence of their letter that greater focus on the specific mechanisms operative in individual patients could lead to better individualization of therapy and better outcomes in ADHF.

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